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Source of dietary lipid may modify the immune response in stressed feeder cattle[1]

T. B. Farran,* C. D. Reinhardt,* D. A. Blasi,* J. E. Minton,* T. H. Elsasser,† J. J. Higgins,‡ and J. S. Drouillard*[2]

*Department of Animal Sciences and Industry, Kansas State University, Manhattan 66506-1600; †USDA, Agricultural Research Service, Beltsville, MD 20705; and ‡Department of Statistics, Kansas State University, Manhattan 66506-1600

ABSTRACT: Five studies were conducted to evaluate the effects of lipid source on performance and health of stressed feeder cattle. A total of 332 heifers (195 ± 2.37 kg initial BW) in trial 1 and 336 heifers (206 ± 1.70 kg initial BW) in trial 2 were fed diets containing ground flaxseed (FLAX), rolled full-fat soybeans (SOY), or tallow (TAL) at 13, 20, or 4%, respectively (DM basis). All diets were formulated to be isonitrogenous and isocaloric. The ADG and G:F for the first 7 d and for the entire feeding period were greater ($P < 0.05$) for TAL and FLAX than for SOY. Percentage of animals treated and retreated for bovine respiratory disease did not differ among dietary treatments. The FLAX treatment increased ($P < 0.05$) total n-3 PUFA concentrations in the plasma, whereas SOY increased ($P < 0.05$) plasma concentrations of total n-6 PUFA. In trial 3, 18 steers were individually fed diets containing TAL and 18 steers were fed a diet containing SOY (20% of DM). In trials 4 and 5, 18 steers were individually fed diets containing TAL and 18 steers were fed diets containing FLAX (12.9% of DM). On d 14 and 17 of study 3, 4, and 5, 16 steers from each dietary treatment were injected i.v. with *Escherichia coli* O55:B5 lipopolysaccharide (LPS), and 2 steers from each diet were injected with saline. Rectal temperatures after LPS challenge were lower ($P < 0.05$) for SOY and FLAX than for TAL, and plasma TNF was greater ($P < 0.05$) for SOY than for TAL. Serum haptoglobin and blood fibrinogen increased and white blood cell count decreased in response to LPS, but none of these variables was affected by treatment. Although this research failed to measure an effect of lipid source on feedlot morbidity or mortality, these studies indicate that altering the source and type of dietary fatty acids may modify the immune response in stressed feeder cattle and that performance may be hindered by feeding full-fat soybeans to receiving cattle.

Key words: endotoxin challenge, immune modulation, lipid, receiving cattle, plasma lipid

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INTRODUCTION

Bovine respiratory disease (**BRD**) has been estimated to be the largest cause of death for all cattle and calves, with a negative annual economic impact of more than $478 million (NASS, 1997). Economic impact of BRD is not confined to death loss because BRD also affects production economics through cost of treatment and pharmaceuticals purchased, loss in feed resources and animal performance from inefficiencies of production in those animals that survive, and increased labor associated with managing morbid animals (Loerch and Fluharty, 1999).

The combination of external stressors such as feed and water deprivation, weaning, climatic changes, commingling, and transport often causes immunosuppression, ultimately resulting in successful colonization by resident viral and bacterial pathogens (Cole, 1996).

Infection of animals by BRD pathogens is correlated with production and release of a variety of inflammatory compounds that often cause a disproportionate and inappropriately activated immune response. This hyperinflammation can result in irreversible lung damage, which compromises disease resistance and productivity of the animal.

Dietary fatty acids have been demonstrated to have immunomodulatory and anti-inflammatory effects in several models of disease in rodents and humans (Calder, 1996). Much of the interest in lipids and their role

[1]Contribution #07-137-J from the Kansas Agric. Exp. Sta., Manhattan 66506.
[2]Corresponding author: jdrouill@ksu.edu
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in immunity has focused on PUFA, specifically the omega-6 (n-6) and omega-3 (n-3) families. Although several studies indicate that dietary n-6 PUFA are proinflammatory, dietary n-3 PUFA ameliorate many inflammatory diseases and have beneficial effects on immune function (Calder, 1998a).

Because nutrition of stressed cattle plays a crucial role in reducing the susceptibility to disease, our objective was to evaluate the differences in growth performance and health status of highly stressed receiving cattle fed diets containing different lipid sources.

MATERIALS AND METHODS

All animals used in the studies reported herein were cared for in compliance with the guidelines established by the Kansas State University Institutional Animal Care and Use Committee.

Trials 1 and 2

Trial 1. Three hundred thirty-two crossbred heifer calves (195 kg initial BW) were used in a 35-d receiving experiment to evaluate growth performance, morbidity, and mortality when calves were fed diets containing different lipid sources. All calves were purchased from sale barns in Kentucky and Tennessee and subsequently shipped to the Beef Cattle Research Center of Kansas State University in September 1999. Upon arrival at the feedlot, calves were allowed ad libitum access to long-stem prairie hay and water and were processed within 24 h. At processing, heifers were individually weighed, ear-tagged, treated for internal and external parasites, administered a zeranol implant (Ralgro, Schering-Plough Animal Health, Kenilworth, NJ), and vaccinated against infectious bovine rhinotracheitis, bovine virus diarrhea, parainfluenza$_3$, and bovine respiratory syncytial virus (BoviShield, Pfizer, New York, NY), and were given a 7-way Clostridial bacterin (Vision 7, Bayer, Shawnee Mission, KS). In addition, the heifers received a prophylactic dose of tilmicosin (0.033 mL/kg of BW; Elanco Animal Health, Greenfield, IN). Calves were allotted in a completely randomized design to their respective dietary treatments and were placed into pens containing 6 to 7 animals. The random assignment to pens was such that loads of cattle were represented equally among treatment groups. Pens (4.3 × 8.5 m) were constructed of metal gates, had solid concrete floors, and were partly covered with open sides. Each dietary treatment was represented by 13 pens per treatment. A second dose of 4-way vaccine (infectious bovine rhinotracheitis, bovine virus diarrhea, parainfluenza$_3$, and bovine respiratory syncytial virus) was administered 7 d after the initial processing, at which time the rectal temperature and BW were recorded.

Heifers were fed diets (Table 1) once daily for 35 d. At initial processing, heifers were randomly allotted to their respective dietary treatments. Treatment diets were based on corn and alfalfa hay, with added tallow (**TAL**; fancy bleachable beef tallow), ground flaxseed (**FLAX**; rich in α-linolenic acid), or rolled full-fat soybeans (**SOY**; rich in linoleic acid). Feed refusals were weighed once weekly, recorded, and discarded so that ad libitum feed intake for each pen could be determined. On d 7 and 35, heifers were individually weighed before feeding (0700 h). Average daily gains and gain efficiencies (G:F, kg of gain/kg of feed) were computed from the initial BW at processing, BW at revaccination, and final BW at d 35.

Health status of all heifers was examined daily. Calves that exhibited clinical signs of BRD, including anorexia, depression, nasal discharge, and rapid or labored breathing, were identified each morning as candidates for antibiotic treatment. Heifers meeting one or more of these criteria were removed from their pens, and their rectal temperatures were measured with an electronic thermometer. Animals were treated for respiratory disease if clinical signs of BRD were accompanied by rectal temperature ≥39.7°C or if they exhibited clinical signs for 2 consecutive days, regardless of the rectal temperature. The initial treatment for BRD was a subcutaneous injection of tilmicosin (0.033 mL/kg of BW). Heifers were returned to their home pen after receiving antibiotic treatment. Calves exhibiting disease symptoms more than 48 h after initial treatment were retreated if signs of clinical illness were accompanied by a rectal temperature of ≥39.7°C. Therapeutic retreatment for BRD consisted of a combination of 0.13 mL/kg of BW of oxytetracycline (Pfizer Animal Health, New York, NY) and 0.11 mL/kg BW of tylosin (Elanco Animal Health).

Trial 2. Three hundred thirty-six crossbred heifer calves (206 kg initial BW) originating from sale barns in the southeastern United States were used for a 35-d receiving trial initiated in October 1999. All heifers were processed and revaccinated as in trial 1. Heifers were randomly allotted to the same dietary treatments as in trial 1.

The first-time therapeutic treatment regimen for BRD was the same as in trial 1, but the protocol for retreatment was modified for trial 2. Heifers were retreated after 48 h of initial treatment if clinical signs of BRD were observed, regardless of the rectal temperature. Therapeutic retreatment for BRD consisted of 0.13 mL/kg of BW of oxytetracycline and 0.11 mL/kg of BW of tylosin.

On the terminal day of trial 2, and at weighing, blood samples were collected via jugular venipuncture on all heifers into 10-mL heparanized Vacutainer tubes (Becton Dickinson, Franklin Lakes, NJ). Tubes were placed immediately on ice until centrifuged at 1,500 × g for 15 min. Plasma was separated and stored frozen until later analysis.

Plasma Analyses. Fatty acids of plasma lipids were converted to methyl esters by a direct transesterification method (Sukhija and Palmquist, 1988). Briefly, 250 μL of plasma was transferred into a 10-mL screw-cap

Table 1. Composition of the diets used in trials 1 through 5 (%, DM basis)[1]

	Trials 1 and 2			Trial 3		Trials 4 and 5	
Item	TAL	FLAX	SOY	TAL	SOY	TAL	FLAX
Ingredient							
Steam-flaked corn	32.9	29.4	32.7			32.9	29.4
Dry-rolled corn							
Alfalfa hay	39.4	39.4	39.5	25.0	25.0	39.4	39.4
Prairie hay				25.0	25.0		
Dehulled soybean meal	15.9	10.5	—	15.9	—	15.9	10.5
Tallow	4.0	—	—	3.8	—	4.0	—
Flaxseed, ground	—	12.9	—			—	12.9
Full-fat soybeans, rolled	—	—	20.0	—	20.0		
Molasses, cane	4.8	4.8	4.8	5.0	5.0	4.8	4.8
Ground corn	2.67	2.67	2.67	2.67	2.67	2.67	2.67
Salt	0.27	0.27	0.27	0.27	0.27	0.27	0.27
Vitamin/mineral premix[2]	0.06	0.06	0.06	0.06	0.06	0.06	0.06
Estimated nutrients[3]							
Trials 1, 3, 4, and 5							
CP	18.4	18.6	18.5	16.8	16.3	18.4	18.6
Ca	0.80	0.80	0.81	0.65	0.64	0.80	0.80
P	0.40	0.42	0.37	0.30	0.30	0.40	0.42
Trial 2							
CP	19.2	19.3	19.0				
Ca	0.66	0.68	0.67				
P	0.33	0.36	0.32				

[1]TAL = tallow; FLAX = ground flaxseed; and SOY = rolled full-fat soybeans.
[2]Provided 2,200 IU of vitamin A, 0.1 mg of Co, 10 mg of Cu, 0.60 mg of I, 60 mg of Mn, 0.1 mg of Se, 60 mg of Zn, and 28 mg of monensin (Elanco Animal Health, Indianapolis, IN) per kilogram of diet DM.
[3]Estimated from analysis of individual feed ingredients.

tube, frozen, and lyophylized. Then, 500 μL of heptadecanoic acid (C17:0; 200 μg/mL in benzene) was added, and the tubes were capped with Teflon-lined caps. The tubes were vortexed to break up the freeze-dried plasma pellet. Next, 2 mL of BF_3-methanol reagent (#33021, Supelco, Bellefonte, PA) was added, and the tubes were swirled gently. The tubes were recapped and subjected to methanolysis in a water bath at 60°C for 60 min. After incubation, the tubes were cooled to room temperature, and 2 mL of water and 0.5 mL of hexane were added to neutralize the mixture, extract the impurities, and increase the volume of the organic phase. The tubes were shaken vigorously and centrifuged at 1,500 × *g* for 7 min. An aliquot of the benzene/hexane upper phase was transferred to a vial containing 3 to 4 small crystals of anhydrous sodium sulfate. Analysis was done using a Hewlett-Packard 5890 gas chromatograph (Palo Alto, CA) fitted with a flame ionization detector and equipped with a Supelcowax-10 capillary column (60 m × 0.32 mm; 0.25-μm film; Supelco). Helium was used as the carrier gas at a flow of 1.4 mL/min. The injection port and detector temperatures were set at 250°C. Column temperature was held at 130°C for 5 min, then raised 3.5°C/min to reach a final temperature of 240°C, which was maintained for 8 min. Peaks were identified by comparison with a standard fatty acid methyl ester mixture (37-component FAME mix, Supelco).

Fatty Acid Composition of Lipid Sources. Fatty acids of lipid ingredients (Table 2) were converted to methyl esters as described by Sukhija and Palmquist (1988). Analysis was performed with a Hewlett-Packard 5890 gas chromatograph equipped with a 2 mm × 2 m glass column packed with 10% SP2330 (Supelco). Nitrogen was used as the carrier gas at a flow of 20 mL/min. The injection port and detector temperatures were set at 225 and 250°C, respectively. Initial column temperature was held at 130°C and raised 3.5°C/min to reach a final temperature of 210°C. Peaks were identified by comparison with a mixture of commercially available fatty acid standards (RM-5 and RM-6, Supelco).

Statistical Analyses. Performance data collected in trials 1 and 2 were analyzed according to the GLM procedure (SAS Inst. Inc., Cary, NC) for a completely randomized design experiment. The model contained effects of dietary treatment. Treatment means were compared by using the PDIFF statement of SAS when protected by a significant *F*-value. Plasma fatty acid concentrations were analyzed as described previously using the MIXED procedure of SAS. Pen of animals served as the experimental unit. Significance was declared at $P \leq 0.05$ and tendencies at $P \leq 0.10$.

Trials 3, 4, and 5

Trial 3. Twenty crossbred beef steers (312 kg of BW) were stratified by BW and randomly assigned, within strata, to 1 of 2 dietary treatments. Dietary treatments (Table 1) consisted of a diet based on corn and soybean meal, with TAL as the added lipid source (rich in saturated and monounsaturated fatty acids), and a diet in which soybean meal and tallow were replaced with SOY

(rich in linoleic acid). Dietary treatments were formulated to be isonitrogenous and isocaloric, and provided approximately equal amounts of vitamins and minerals. Steers were housed in individual pens, with drinking water available at all times, and were fed their respective diets once daily for a 14-d acclimation period. On d 14, all steers were fitted with jugular catheters, and 8 steers from each dietary treatment were injected intravenously with bacterial endotoxin (0.2 μg/kg of BW *E. coli* O55:B55 lipopolysaccharide, Sigma Chemical Company, St. Louis, MO). Two steers from each dietary treatment were injected intravenously with saline to establish baseline blood measurements and temperature readings. Blood samples (via jugular catheter) were obtained immediately before (0 h), and at 2, 3, 4, 5, and 24 h after LPS or saline injection.

Rectal temperatures and infrared body surface temperatures were recorded immediately before (0 h), and at 1, 2, 3, 4, and 6 h after LPS or saline injection. Body surface temperatures were recorded with imaging equipment that consisted of a high-resolution, shortwave (3 to 5 μm), cooled, radiometric, infrared, thermal imaging camera (ThermaCam 280, Inframetrics, North Billerica, MA). The camera was equipped with a 16° field of view lens, with the images displayed in a 256 × 256-pixel, focal plane array. The left side of each steer was imaged from a distance of 1.8 m. Images were stored digitally on a PCMCIA memory card. Postimaging analysis of captured images was performed with Thermagram 95 (Inframetrics) software. The software allowed adjustment for variances in ambient temperature, relative humidity, and wind speed.

The LPS challenge procedure was repeated on d 17 (3 d after the initial LPS or saline injection) to further evaluate the effects of dietary lipid on response to endotoxin. Animals that received the LPS or saline injection on d 14 were readministered the same injection. Blood samples on d 17 were taken immediately before (0 h), and at 1, 2, 3, 4, 6, and 24 h after intravenous injection of LPS or saline. Temperature readings were taken at 0, 1, 2, 3, 4, and 6 h after injection.

Blood samples were collected into Vacutainer tubes containing EDTA (Becton Dickinson, Franklin Lakes, NJ). Tubes were immediately placed on ice and kept cold until centrifuged at 1,500 × *g* for 15 min. Plasma was separated from each tube, transferred to a collection vial, and stored frozen. A fourth Vacutainer tube containing no anticoagulant was allowed to clot for at least 30 min at ambient temperature and was centrifuged at 1,500 × *g* for 15 min. Serum was separated, transferred to a microcentrifuge tube, and stored frozen.

Blood plasma was analyzed for concentrations of prostaglandin E_2 (**PGE**) and tumor necrosis factor-α (**TNF**). Whole blood was measured for total white blood cell count (**WBC**) and concentrations of fibrinogen (**FIB**). Blood serum was analyzed for concentrations of haptoglobin (**HAP**).

Table 2. Long-chain fatty acid concentrations in dietary lipid ingredients used in trials 1 and 2

	Ingredient[1]		
Item	TAL	FLAX	SOY
Total fatty acids, % of DM	79.5	34.8	18.3
Fatty acid	% of total fatty acids		
C14:0	3.2	—	—
C16:0	24.9	6.4	12.3
C16:1	3.2	—	—
C18:0	22.5	3.1	4.1
C18:1	43.6	20.3	21.5
C18:2	2.3	15.9	54.0
C18:3	0.3	54.2	8.0
C20:5	—	—	—
C22:5n6	—	—	—
C22:6n3	—	—	—

[1]TAL = tallow; FLAX = ground flaxseed; and SOY = rolled full-fat soybeans.

Trial 4. Eighteen crossbred beef steers (354 kg) were used in a randomized complete-block design experiment to evaluate response to LPS challenge when steers were fed diets containing each of 3 lipid sources. Dietary treatments (Table 1) contained TAL or FLAX (rich in α-linolenic acid). Steers were housed under the same conditions as in trial 3 and were fed their respective dietary treatments once daily for a 14-d acclimation period.

On d 14, all steers were fitted with jugular catheters before administration of LPS. A saline solution containing LPS (0.2 μg/kg of BW *E. coli* O55:B55 lipopolysaccharide, Sigma Chemical Company) was injected into the catheters, which were subsequently flushed with a heparanized saline solution. Blood samples were obtained immediately before (0 h), and at 1, 2, 3, 4, 6, and 24 h after LPS challenge. The procedure was repeated on d 17 (3 d after the initial LPS challenge).

Rectal temperatures and infrared body surface temperatures on all steers were recorded as in trial 3, except that the infrared camera was positioned 3.6 m above the steers to obtain a dorsal image. Temperature readings were recorded immediately before (0 h), then at 1, 2, 3, 4, 5, and 6 h after LPS injection for both d 14 and 17 injections of LPS.

Trial 5. Sixty days after the end of trial 4, the endotoxin challenge experiment was repeated using the same experimental treatments and animals. Steers were reweighed (421 kg of BW) and again stratified by BW and randomly allocated, within strata, to each of the 3 dietary treatments. All other procedures were performed as in trial 4.

Statistical Analyses. Blood parameters and temperature profiles were analyzed as a randomized complete block design with repeated measures using to the MIXED procedure of SAS. Animal served as the experimental unit. The model contained effects of dietary treatment, time, and the treatment × time interaction. Animal and animal × treatment were included as ran-

Table 3. Effects of lipid source on performance of feeder heifers in trials 1 and 2

Item[1]	Diet[1] TAL	FLAX	SOY	SEM
Trial 1				
No. of pens (heifers)	13 (83)	13 (83)	13 (83)	
Initial BW, kg	194.9	190.9	197.6	2.37
End BW, kg	239.2	236.7	230.2	3.67
DMI, kg/d	4.74[a]	4.72[a]	4.10[b]	0.204
ADG, d 1 to 7, kg	0.770[a]	0.815[a]	0.274[b]	0.172
ADG d 7 to 35, kg	1.38[a]	1.42[a]	1.11[b]	0.196
ADG, d 1 to 35, kg	1.24[a]	1.28[a]	0.92[b]	0.075
G:F, kg/kg	0.262[a]	0.266[a]	0.222[b]	0.010
First time treatment, %	50.0	51.1	53.5	6.20
Second time treatment, %	29.1	18.7	27.3	5.15
Mortality, %	4.6	2.4	1.3	2.47
Trial 2				
No. of pens (heifers)	13 (84)	13 (84)	13 (84)	
Initial BW, kg	206.7	205.2	206.9	1.701
End BW, kg	249.1[a]	250.6[a]	239.5[b]	2.52
DMI, kg/d	4.70[ab]	4.81[a]	4.33[b]	0.116
ADG, d 1 to 7, kg	0.386[a]	0.605[a]	–0.055[b]	0.170
ADG, d 7 to 35, kg	1.41[a]	1.48[a]	1.20[b]	0.126
ADG, d 1 to 35, kg	1.20[a]	1.30[a]	0.95[b]	0.055
G:F, kg/kg	0.256[a]	0.270[a]	0.217[b]	0.009
First time treatment, %	70.9	72.9	68.9	4.26
Second time treatment, %	37.7	40.8	38.5	6.29
Mortality, %	1.2	1.2	0.0	0.91

[a,b]Within a row, means without a common superscript letter differ ($P < 0.05$).
[1]TAL = tallow; FLAX = ground flaxseed; and SOY = rolled full-fat soybeans.

dom variables to serve as main plot and subplot error terms, respectively. In the absence of a treatment × challenge interaction, effectiveness of the challenge was evaluated by comparing least squares means between the main effects of all animals challenged with LPS and those injected with saline alone. A *t*-test for all possible pair-wise comparisons (PDIFF option) among means was done when protected by an overall significant F-test. Significance was declared at $P < 0.05$, and tendencies were declared at $P < 0.10$.

RESULTS

Trial 1

Heifer performance during trial 1 is summarized in Table 3. Daily gain was decreased ($P < 0.05$) for animals consuming the SOY diet, compared with those animals consuming the TAL and FLAX diets during the first week after arrival, wk 2 through 5, and for the entire receiving period. Gains of cattle fed FLAX or TAL were not different throughout the entire 35-d receiving period but were greater ($P < 0.05$) than those of heifers fed SOY. There was a diet effect on DMI (4.74, 4.72, and 4.10 kg/d for TAL, FLAX, and SOY, respectively) throughout the entire receiving period. Heifers consuming the TAL and FLAX diets consumed more feed ($P < 0.05$) than heifers on the SOY diet. Efficiencies of gain were greater ($P < 0.05$) for animals fed TAL and FLAX diets than for heifers fed SOY (0.262, 0.266 vs. 0.222 kg of gain/kg of feed, respectively).

To ensure a natural challenge and to more effectively elucidate any potential effects of dietary lipid source in truly morbid calves, relatively high-risk calves were procured for this study, as demonstrated by the high morbidity rates. Antibiotic therapy for first-time treatment of BRD, while relatively high, was not different among dietary treatments, with heifers fed TAL, FLAX, and SOY diets having 50.0, 51.1, and 53.5% morbidity, respectively. Fewer animals ($P = 0.16$) consuming the FLAX diet required antibiotic retreatment for BRD, compared with those fed TAL, but significant treatment differences were not detected (29.1, 18.7, and 27.3% second time re-treatment for TAL, FLAX, and SOY, respectively). Likewise, mortality due to BRD was not different between dietary treatments (4.6, 2.4, and 1.3% for TAL, FLAX, and SOY, respectively).

Trial 2

Heifer performance during trial 2 followed the same pattern as in trial 1 and is summarized in Table 3. Daily gain was decreased ($P < 0.05$) for animals consuming the SOY diet, compared with those animals consuming the TAL and FLAX diets during the first week after arrival, wk 2 through 5, and for the entire receiving period. Over the entire 35-d receiving period, ADG was greater ($P < 0.05$) for heifers fed the TAL and FLAX diets than for those fed the SOY diet (1.29 and 1.30 vs. 0.95 kg, respectively). Similar to trial 1, average daily DMI during trial 2 was affected by diet. Heifers allotted to the FLAX diet consumed the most feed, whereas heifers

Table 4. Long-chain fatty acid concentrations in plasma, trial 2

Fatty acid	Diet[1] TAL	FLAX	SOY	SEM
	μg of fatty acid/mL of plasma			
C14:0	15.3	12.8	12.1	1.17
C15:0	10.8[a]	9.7[a]	8.5[b]	0.49
C16:0	194.4[a]	157.0[b]	170.2[b]	6.46
C16:1	28.9[a]	22.9[c]	14.4[d]	1.47
C17:1	10.7[a]	4.3[b]	3.5[b]	1.04
C18:0	259.7[ac]	304.8[b]	278.6[bc]	9.78
C18:1n9	228.7[a]	150.3[b]	122.5[c]	5.86
C18:2n6	544.2[a]	609.6[a]	698.0[b]	26.33
C18:3n6	13.7[a]	11.6[ab]	11.3[b]	0.78
C18:3n3	53.9[a]	262.7[b]	46.7[a]	4.90
C20:3n6	23.9[a]	16.3[c]	25.0[a]	1.12
C20:3n3	33.2[a]	28.0[b]	39.2[c]	1.43
C20:5n3	14.4[a]	28.7[c]	13.3[a]	1.07
C22:6n3	8.9[a]	9.3[a]	8.0[a]	0.96
Total (n-3)	110.2[a]	335.2[b]	112.4[a]	7.58
Total (n-6)	601.7[a]	625.6[a]	770.6[b]	34.80

[a–c]Within a row, means without a common superscript letter differ ($P < 0.05$).

[1]TAL = tallow; FLAX = ground flaxseed; and SOY = rolled full-fat soybeans.

consuming the SOY diet consumed the least (4.81 vs. 4.33 kg/d; $P < 0.05$). However, ADFI was not different for TAL (4.70 kg/d), compared with FLAX or SOY. Efficiencies of gain were greater ($P < 0.05$) for animals fed TAL and FLAX diets, compared with those fed SOY (0.256 and 0.270 vs. 0.217 kg of gain/kg of feed, respectively).

Fewer animals died due to respiratory disease during trial 2 than during trial 1 (Table 3), but cattle had an overall greater incidence of first-time antibiotic treatment for BRD than in trial 1. The minimum threshold rectal temperature to qualify for antibiotic retreatment was eliminated in trial 2, and heifers were retreated solely if clinical symptoms (inappetance, depression, nasal discharge) were exhibited 48 h or more after initial treatment. Retreatment rates were not affected by diet (37.7, 40.8, and 38.5% for TAL, FLAX, and SOY, respectively).

Fatty acid profiles of plasma lipids are shown in Table 4. Dietary treatments exhibited profound effects on plasma long-chain fatty acids. Other researchers have shown similar effects of dietary lipid manipulation on plasma lipids in rodents (Mascioli et al., 1989; De Schrijver et al., 1991), humans (Marangoni et al., 1993), nonhuman primates (McMurchie et al., 1990), and in serum fatty acids of ruminants (Marchello et al., 1972; Ashes et al., 1992).

Total plasma lipids from heifers fed FLAX exhibited enrichments ($P < 0.05$) in α-linolenic (C18:3n3) and eicosapentaenoic (C20:5n3) acids, and a concomitant decrease in dihomo-γ-linolenic acid (C20:3n6), compared with lipids of cattle fed the TAL and SOY diets. Heifers fed SOY had greater ($P < 0.05$) proportions of linoleic acid (C18:2n6) compared with animals receiving all other dietary treatments. Moreover, total n-3 concentrations of plasma lipids were greater ($P < 0.05$) in animals fed FLAX than in those fed TAL or SOY. Plasma of heifers fed SOY had larger ($P < 0.05$) amounts of n-6 PUFA than those fed TAL or FLAX. Calves receiving the TAL diet had greater ($P < 0.05$) proportions of C16:0, C17:1, and C18:1n9 in plasma lipids than heifers fed FLAX or SOY. Furthermore, plasma from heifers fed the TAL diet contained more ($P < 0.05$) C18:3n6 than heifers fed the SOY diet, but C18:3n6 content was not different from that of heifers fed FLAX.

Trial 3

Injection of LPS resulted in dramatic increases in rectal temperature, TNF and HAP, and in reductions in WBC (Tables 5 and 6). Rapid changes in rectal temperatures, as well as changes in WBC, TNF, and HAP, relative to saline-injected animals, indicate that the model was effective in stimulating an immune response.

There was a treatment × time interaction ($P < 0.01$) for rectal temperature and TNF-α, but not for the other variables. The post-challenge increase in rectal temperature was greater for cattle fed TAL than for those fed SOY after LPS injection on both d 14 and 17 (Table 6). After the first LPS injection (d 14), rectal temperatures at 3 h were greater ($P < 0.05$) for TAL-fed animals than for those fed SOY and tended ($P < 0.10$) to be greater at 4 h. Likewise, d 17 injection of LPS resulted in a tendency for animals fed TAL to exhibit greater ($P < 0.10$) rectal temperatures at 1 h than those of cattle fed SOY. Body surface temperatures were not different for SOY and TAL treatments after either LPS injection.

Rapid increases in plasma TNF amounts were evident immediately after LPS injection compared with the saline controls and returned to baseline amounts after 3 h (Figures 1 and 2). Plasma TNF was greater for animals fed SOY than for animals fed TAL after both injections of LPS. Plasma TNF after the d 14 challenge was greater ($P < 0.01$) at 2 h for animals fed SOY than for those fed TAL. Likewise, TNF after d 17 challenge was greater ($P < 0.05$) for animals fed SOY than for cattle fed TAL at 1 and 2 h after LPS injection. These results contrast the rectal temperature results, which indicated a greater inflammatory response for TAL vs. SOY. However, TNF concentrations following the d-17 challenge were not affected by dietary lipid source.

Peripheral blood WBC count was significantly reduced within 2 h after LPS injection; by 24 h the reduction was stabilized, and WBC returned to baseline (Table 5). No differences among dietary treatments were observed. Serum HAP concentrations increased in response to LPS, but were not different between diets. Concentrations of PGE were highly variable and not different among treatments.

Table 5. Blood constituents postchallenge, trial 3[1]

	h after LPS injection								
Item	0	1	2	3	4	5	6	24	SEM
d 14 challenge									
Fibrinogen, mg/dL									
TAL-LPS[1]	438	—	463	438	450	338	—	538	53.4
SOY-LPS[1]	350	—	300	325	288	263	—	425	53.4
TAL-NO LPS	400	—	400	400	350	300	—	350	106.7
SOY-NO LPS	350	—	400	400	300	200	—	300	106.7
Total WBC,[2] $\times 10^3$/mL									
TAL-LPS	9.9	—	2.5	1.7	2.4	3.2	—	10.6	0.44
SOY-LPS	10.5	—	2.4	1.5	1.7	2.4	—	10.5	0.44
TAL-NO LPS	9.1	—	8.7	8.6	8.4	9.0	—	9.0	0.88
SOY-NO LPS	9.6	—	9.2	8.9	9.2	8.9	—	8.8	0.88
Prostaglandin E_2, ng/mL									
TAL-LPS	595	—	701	490	529	576	—	430	61.4
SOY-LPS	545	—	650	410	566	612	—	387	61.4
TAL-NO LPS	756	—	615	513	690	680	—	442	122.9
SOY-NO LPS	663	—	467	527	547	458	—	385	122.9
Haptoglobin,[2] mg %									
TAL-LPS	7.9	—	9.6	12.4	13.3	8.9	—	27.9	0.98
SOY-LPS	7.5	—	9.0	12.8	12.8	9.6	—	29.1	0.92
TAL-NO LPS	7.5	—	8.5	12.0	13.0	9.0	—	10.0	1.84
SOY-NO LPS	10.5	—	9.0	13.0	13.0	9.5	—	10.5	1.84
d 17 challenge									
Fibrinogen, mg/dL									
TAL-LPS	400	386	443	443	414	—	429	343	46.2
SOY-LPS	450	425	450	425	400	—	388	300	43.2
TAL-NO LPS	350	350	400	350	250	—	350	400	86.4
SOY-NO LPS	350	400	350	450	300	—	250	400	86.4
Total WBC,[2] $\times 10^3$/mL									
TAL-LPS	8.7	3.1	2.8	2.3	3.0	—	4.8	8.9	0.43
SOY-LPS	8.6	2.7	2.3	1.8	2.3	—	4.0	9.7	0.40
TAL-NO LPS	7.5	7.9	7.7	7.3	8.7	—	8.7	8.7	0.80
SOY-NO LPS	9.3	8.9	9.4	10.6	11.1	—	11.2	10.5	0.80
Prostaglandin E_2, ng/mL									
TAL-LPS	421	553	496	746	751	—	761	712	75.9
SOY-LPS	418	507	476	595	624	—	758	652	71.0
TAL-NO LPS	506	654	599	793	794	—	1,006	344	142.0
SOY-NO LPS	414	337	435	820	689	—	810	1,017	142.0
Haptoglobin, mg %									
TAL-LPS	16.5	16.8	15.0	14.8	12.5	—	12.3	18.7	4.07
SOY-LPS	18.6	20.0	21.1	21.6	18.5	—	18.6	20.9	3.52
TAL-NO LPS	8.5	8.0	8.5	9.5	8.5	—	10.5	9.5	7.05
SOY-NO LPS	7.0	8.0	9.5	11.5	9.0	—	9.5	10.0	7.05

[1]TAL = tallow; SOY = rolled full-fat soybeans; and LPS = lipopolysaccharide.
[2]Contrast, LPS vs. NO-LPS ($P < 0.05$).

Trial 4

Injection of the bacterial LPS resulted in dramatic changes in rectal temperature, TNF, PGE, acute-phase proteins (HAP and FIB), and WBC count (Tables 7 and 8). Changes in rectal temperature after LPS administration were greatest for TAL and lowest for FLAX during the d 14 LPS challenge (Figure 3). On d 14, rectal temperatures for cattle fed TAL were greater ($P < 0.05$) at 3, 4, 5, and 6 h after LPS injection, when compared with temperatures for cattle fed FLAX (Figure 3).

Rapid increases in TNF production (Table 7) were evident by 2 h after both LPS injections, and decreased to prechallenge amounts by 4 h. Peripheral blood WBC count fell immediately after LPS injection on both d 14 and 17. The WBC count returned to pre-LPS injection values by 24 h. This observation is in agreement with trial 3, in which LPS reduced WBC count, compared with that in non-LPS injected control animals. Dietary treatments did not cause differences in WBC count after LPS injection.

Trial 5

In agreement with trials 3 and 4, results observed in trial 5 showed that injection of cattle with LPS resulted in changes in rectal temperature, TNF, PGE, acute-phase proteins (HAP and FIB), and WBC count (Tables 9 and 10).

Table 6. Temperature profiles, trial 3[1]

Item	h after LPS injection 0	1	2	3	4	6	SEM
d 14 challenge							
Rectal temperature,[2] °C							
TAL-LPS	38.97	39.76	40.11	40.47[a]	40.39[c]	39.53	0.192
SOY-LPS	38.97	39.63	39.71	39.86[b]	39.91[d]	39.52	0.192
TAL-NO LPS	39.22	38.70	38.81	38.81	38.67	38.81	0.384
SOY-NO LPS	38.94	39.03	38.92	39.03	38.72	38.67	0.384
Body surface temperature, °C							
TAL-LPS	21.4	22.3	22.3	22.7	22.4	22.3	0.51
SOY-LPS	22.3	22.3	22.8	22.5	22.1	22.2	0.51
TAL-NO LPS	20.7	21.3	21.6	22.2	21.2	20.1	1.01
SOY-NO LPS	24.3	24.8	24.8	24.7	23.9	23.7	1.01
d 17 challenge							
Rectal temperature,[2] °C							
TAL-LPS	39.48	40.24[c]	40.31	40.57	40.39	39.26	0.136
SOY-LPS	39.50	39.92[d]	40.03	40.31	40.42	39.25	0.127
TAL-NO LPS	39.33	39.36	39.08	39.25	38.86	38.81	0.254
SOY-NO LPS	39.61	39.78	40.22	39.86	39.58	39.03	0.254
Body surface temperature, °C							
TAL-LPS	25.2	24.5	25.5	25.4	25.8	25.1	0.49
SOY-LPS	25.2	24.4	25.1	25.1	25.4	24.9	0.46
TAL-NO LPS	24.4	24.5	24.9	24.3	23.8	22.7	0.91
SOY-NO LPS	25.9	26.5	26.8	26.2	25.5	25.3	0.91

[a,b]Within a day and temperature measurement, TAL differed from SOY ($P < 0.05$).
[c,d]Within a day and temperature measurement, TAL tended to be different from SOY ($P < 0.10$).
[1]TAL = tallow; SOY = rolled full-fat soybeans; and LPS = lipopolysaccharide.
[2]Contrast, LPS vs. NO-LPS ($P < 0.05$).

Dietary effects on rectal temperature were not evident after injection of LPS. There was an effect of LPS injection on plasma TNF concentration. Plasma concentrations of TNF peaked and returned to baseline by 4 h after LPS injection on both d 14 and 17, but a treatment × time interaction was not detected, indicating that dietary treatments did not influence TNF concentration after LPS injection.

In accordance with observations from trial 4, trial 5 showed peripheral blood WBC numbers were significantly reduced by 1 h after LPS injection; by 24 h the reduction was stabilized, and WBC returned to baseline amounts.

Plasma PGE concentrations were highly variable and not different among cattle fed the different experimental diets. This observation further supports our speculation that PGE is difficult to accurately measure in vivo.

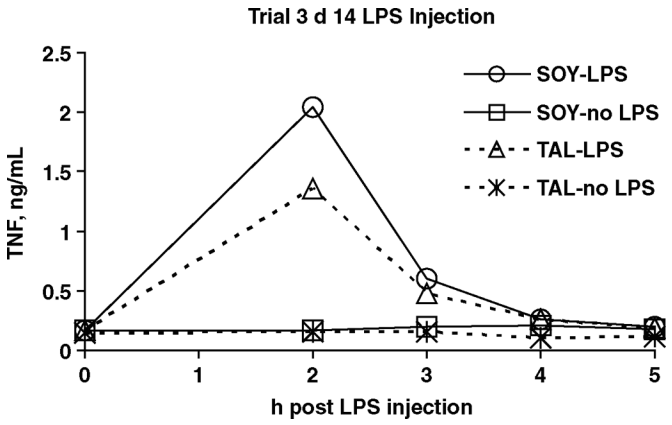

Figure 1. Effect of lipopolysaccharide (LPS) injection on tumor necrosis factor α (TNFα) production vs. time for steers fed tallow (TAL) or full-fat soybeans (SOY) in trial 3. TAL-LPS differed from SOY-LPS at h 2 ($P < 0.01$; treatment × time interaction, $P < 0.01$).

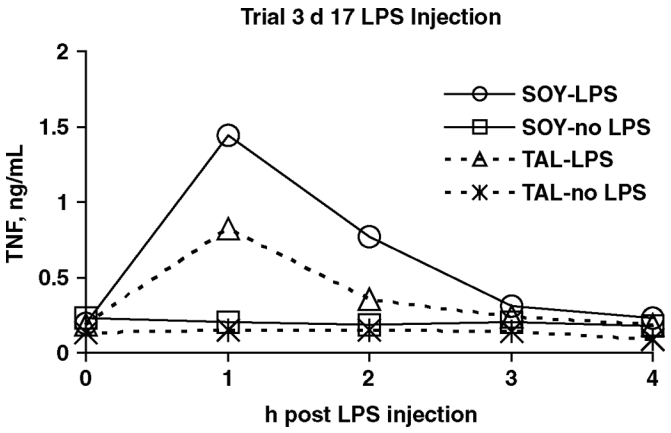

Figure 2. Effect of lipopolysaccharide (LPS) injection on tumor necrosis factor α (TNFα) production vs. time for steers fed tallow (TAL) or full-fat soybeans (SOY) in trial 3. TAL-LPS differed from SOY-LPS at h 1 and 2 ($P < 0.05$; treatment × time interaction, $P < 0.01$).

Table 7. Blood constituents, trial 4[1]

Item	h after LPS injection 0	1	2	3	4	6	24	SEM
d 14 challenge								
Tumor necrosis factor-α, ng/mL								
TAL	0.17[a]	0.88[c]	0.51[b]	0.24[ab]	0.20[a]	—	—	0.133
FLAX	0.19[a]	0.79[c]	0.64[bc]	0.34[ab]	0.19[a]	—	—	0.146
Fibrinogen, mg/dL								
TAL	350[ab]	350[ab]	317[ab]	300[a]	267[a]	317[ab]	400[b]	47.6
FLAX	350[bc]	350[bc]	317[abc]	250[a]	300[ab]	333[abc]	400[c]	47.6
Total WBC, 10^3/mL								
TAL	9.7[a]	2.8[b]	3.1[b]	2.7[b]	3.3[b]	5.0[ab]	9.7[a]	0.88
FLAX	10.5[a]	3.1[b]	3.7[b]	3.6[b]	3.8[b]	6.3[ab]	10.3[a]	0.88
Prostaglandin E_2, ng/mL								
TAL	824[a]	852[a]	950[ac]	888[a]	623[b]	735[ab]	578[b]	97.8
FLAX	675[a]	775[ab]	878[b]	732[ab]	624[a]	638[a]	627[a]	89.3
Haptoglobin, mg %								
TAL	5.2[a]	5.7[a]	5.8[a]	5.3[a]	5.5[a]	8.0[ab]	10.8[b]	2.33
FLAX	8.8[ab]	8.7[ab]	8.5[ab]	8.7[ab]	9.0[a]	9.0[a]	14.8[b]	2.33
d 17 challenge								
Tumor necrosis factor-α, ng/mL								
TAL	0.13[a]	0.60[c]	0.31[ab]	0.19[a]	0.17[a]	—	—	0.063
FLAX	0.15[a]	0.46[c]	0.27[ab]	0.18[a]	0.13[a]	—	—	0.069
Fibrinogen, mg/dL								
TAL	350[ab]	333[a]	333[a]	283[a]	317[ab]	317[ab]	467[b]	39.3
FLAX	300[a]	383[b]	367[ab]	383[b]	350[ab]	383[b]	400[b]	39.3
Total WBC, × 10^3								
TAL	8.7[a]	2.9[b]	3.2[b]	2.9[b]	3.3[b]	5.0[c]	8.1[a]	0.55
FLAX	9.6[a]	3.1[b]	3.7[b]	3.5[b]	4.0[b]	5.7[c]	8.3[d]	0.55
Prostaglandin E_2, ng/mL								
TAL	694[a]	508[b]	474[c]	329[c]	336[cd]	507[b]	610[a]	59.0
FLAX	694[a]	417[c]	397[c]	338[c]	292[c]	411[c]	554[ab]	53.9
Haptoglobin, mg %								
TAL	6.7[ab]	6.3[a]	9.3[bc]	9.5[c]	9.2[bc]	10.7[c]	15.0[d]	1.20
FLAX	9.5[ab]	8.7[a]	10.8[ab]	10.5[ab]	10.5[b]	11.2[ab]	9.2[ab]	1.20

[a–d]Within a row, means without a common superscript letter differ ($P < 0.05$).
[1]LPS = lipopolysaccharide; TAL = tallow; and FLAX = flaxseed.

DISCUSSION

In general, our results indicate that dietary lipid source had marked effects on plasma fatty acids. Inclusion in the diet of flaxseed increased the proportion of n-3 PUFA, whereas feeding cattle full-fat soybeans increased the proportion of n-6 PUFA, and these results were parallel to the types of fatty acids present in these feed sources (Table 2).

Many factors can affect hydrogenation of PUFA in ruminant diets, including the nature and amount of dietary lipid fed, the type of protective treatment, and

Table 8. Temperature profiles, trial 4[1]

Item	h after LPS injection 0	1	2	3	4	5	6	SEM
d 14 challenge								
Body surface temperature, °C								
TAL	32.9	32.4	32.1	31.3	31.9	30.1	29.0	0.48
FLAX	33.4	31.1	32.3	31.7	31.0	29.7	28.5	0.48
d 17 challenge								
Rectal temperature, °C								
TAL	39.1	39.6	39.4	39.7	39.4	39.2	39.2	0.15
FLAX	38.9	39.4	39.4	39.3	39.2	39.0	38.7	0.17
Body surface temperature, °C								
TAL	34.0	32.5	31.9	29.4	28.7	27.5	26.5	0.36
FLAX	34.0	32.6	32.1	30.6	28.4	27.2	26.9	0.36

[1]LPS = lipopolysaccharide; TAL = tallow; and FLAX = flaxseed.

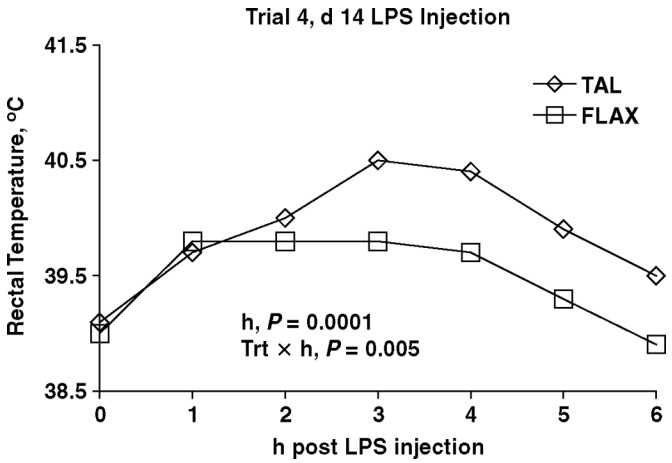

Figure 3. Effect of LPS injection on rectal temperature response vs. time for steers fed different lipid sources in trial 4. Tallow (TAL) was greater than flaxseed (FLAX) at h 3, 4, 5, and 6 ($P < 0.05$; treatment × time interaction, $P < 0.01$).

the nature and amount of forages and concentrates included in the diet. In turn, all of these will influence the rumen microbial ecosystem (lipolytic and biohydrogenating organisms) and the transit rate of digesta through the rumen, thus allowing escape of a certain amount of PUFA from lipolysis and subsequent hydrogenation to successfully bypass the rumen (Palmquist and Jenkins, 1980; Chilliard, 1993).

The responses of TNF to LPS challenge in the present studies are in agreement with Ohtsuka et al. (1997), who observed increased circulating TNF in adult cows injected with LPS, compared with TNF of saline controls. Other researchers have demonstrated increased and decreased TNF concentrations in animals fed n-3-PUFA enriched diets. Chang et al. (1992) injected mice with LPS and observed a marked increase ($P < 0.005$) in serum TNF for mice fed n-3 enriched fish oil compared with those fed corn, coconut oil, or a low-fat diet. Furthermore, they reported that peritoneal macrophages in mice fed fish oil exhibited an exaggerated in vitro TNF release when exposed to LPS. These findings are also in agreement with those of Lokesh et al. (1990). Contrary to these findings, Endres et al. (1989) observed that supplementation of the diet with fish oil concentrate [rich in eicosapentaenoic acid (**EPA**) and

Table 9. Blood constituents, trial 5[1]

	h after LPS injection							
Item	0	1	2	3	4	6	24	SEM
d 14 challenge								
Tumor necrosis factor-α, ng/mL								
TAL	0.19[a]	3.97[c]	2.22[b]	0.59[a]	0.36[a]	—	—	0.392
FLAX	0.20[a]	3.15[c]	2.00[b]	0.66[a]	0.35[a]	—	—	0.392
Fibrinogen, mg/dL								
TAL	250[a]	267[a]	283[a]	283[a]	300[a]	317[a]	417[b]	37.9
FLAX	283[a]	283[a]	333[ab]	267[a]	267[a]	333[ab]	383[b]	37.9
Total WBC, × 103/mL								
TAL	8.6[a]	2.4[bd]	1.8[bc]	1.4[c]	1.6[bc]	2.7[bd]	10.8[e]	0.49
FLAX	7.9[a]	2.3[bcd]	1.4[c]	1.3[c]	1.6[bc]	2.9[bd]	9.8[e]	0.49
Prostaglandin E_2, ng/mL								
TAL	278[a]	352[a]	523[b]	620[b]	577[b]	624[b]	604[b]	85.7
FLAX	333[a]	364[ab]	576[c]	501[bc]	488[bc]	524[c]	574[c]	78.2
Haptoglobin, mg %								
TAL	5.7[ab]	5.7[ab]	7.0[ab]	7.7[b]	6.5[ab]	4.5[a]	18.7[c]	1.14
FLAX	6.2[ab]	6.0[ab]	5.7[ab]	7.0[b]	6.8[ab]	4.7[a]	16.3[c]	1.14
d 17 challenge								
Tumor necrosis factor-α, ng/mL								
TAL	0.17[a]	0.98[c]	0.38[b]	0.22[a]	0.23[a]			0.078
FLAX	0.17[a]	0.80[c]	0.32[a]	0.23[a]	0.24[a]			0.078
Total WBC, × 103								
TAL	8.9[a]	2.7[b]	2.4[b]	2.4[b]	2.7[b]	4.6[c]	7.8[d]	0.47
FLAX	8.0[a]	2.5[b]	2.1[b]	2.1[b]	2.6[b]	4.4[c]	6.9[d]	0.47
Fibrinogen, mg/dL								
TAL	417[abd]	433[a]	367[ab]	300[c]	417[ad]	467[d]	350[bc]	38.6
FLAX	417[ab]	433[a]	400[ab]	383[ab]	350[b]	433[a]	400[ab]	38.6
Prostaglandin E_2, ng/mL								
TAL	311[ab]	319[ab]	459[bc]	513[c]	286[a]	349[ab]	458[bc]	76.2
FLAX	280	320	307	303	212	246	281	69.6
Haptoglobin, mg %								
TAL	10.8	10.5	10.3	8.8	9.7	10.2	13.3	4.97
FLAX	15.3[ab]	16.7[b]	14.0[ab]	13.5[ab]	14.3[ab]	10.7[a]	13.2[ab]	4.97

[a–e]Within a row, means without a common superscript letter differ ($P < 0.05$).
[1]LPS = lipopolysaccharide; TAL = tallow; and FLAX = flaxseed.

Table 10. Temperature profiles, trial 5[1]

	h after LPS injection							
Item	0	1	2	3	4	5	6	SEM
d 14 challenge								
Rectal temperature, °C								
TAL	38.6[a]	39.2[b]	39.6[c]	40.1[d]	40.1[d]	39.6[c]	39.0[ab]	0.16
FLAX	38.9[a]	39.5[bc]	39.8[c]	40.6[d]	40.8[d]	39.8[c]	39.2[ab]	0.16
Body surface temperature, °C								
TAL	18.0	17.4	22.0	20.4	17.8	17.3	16.6	0.84
FLAX	17.7	16.4	20.6	19.6	18.4	17.3	16.2	0.84
d 17 challenge								
Rectal temperature, °C								
TAL	38.7[a]	39.1[b]	39.3[b]	39.7[c]	39.5[c]	39.2[b]	39.1[ab]	0.14
FLAX	38.9[a]	39.3[b]	39.5[bc]	39.7[c]	39.4[b]	39.2[b]	39.1[ab]	0.14
Body surface temperature, °C								
TAL	11.8	11.1	11.1	10.6	10.5	11.7	11.3	0.83
FLAX	11.5	9.9	10.5	10.1	9.7	10.8	10.7	0.83

[a–d]Within a row, means without a common superscript letter differ ($P < 0.05$).
[1]LPS = lipopolysaccharide; TAL = tallow; and FLAX = flaxseed.

docosahexaenoic acid] resulted in reduced ability of human monocytes to produce TNF after stimulation with endotoxin. Yaqoob and Calder (1995) fed mice diets that contained 25 g/kg (low-fat) or 200 g/kg of hydrogenated coconut, olive, safflower, or fish oil for 8 wk, and reported decreased production of TNF ex vivo by peritoneal macrophages from mice fed n-3 PUFA. Engelberts et al. (1993) showed that consumption of palm oil (rich in saturated and monounsaturated fatty acids) reduced the ability of human peripheral blood cells to produce TNF in response to LPS challenge. Likewise, Calder (1997) summarized numerous research trials and concluded that feeding laboratory animals n-3 PUFA-rich diets resulted in decreased production of TNF-α by macrophages.

The blunted febrile response observed in the animals fed FLAX rather than TAL may be partly attributed to the type of eicosanoids formed from the inflammatory cells of animals consuming these n-3 PUFA-enriched diets. Other researchers (Pomposelli et al., 1989) have shown that diets containing n-3-enriched fish oil reduce the fever response in guinea pigs. Docosahexaenoic and α-linolenic acids can be converted to EPA in animal cells (Calder, 1996). Eicosapentaenoic acid, by virtue of its ability to compete with arachidonic acid, can competitively inhibit production of eicosanoids such as the 2-series prostaglandins and the 4-series leukotrienes from arachidonic acid (Calder, 1998b); thereby reducing inflammation. These eicosanoids from arachidonic acid metabolism are much more proinflammatory and biologically active than those from EPA (Calder, 1997). Differences in dietary PUFA intake have been shown to alter plasma (Pomposelli et al., 1989; McMurchie et al., 1990; De Schrijver et al., 1991) and immune cell membrane composition (Lee et al., 1985) in rodents and monkeys. Because dietary fat can alter cell membrane composition, it is a major modulator of immune function (Grimble, 1994). Our study suggests that n-3 PUFA in the diet may be anti-inflammatory with regard to the febrile response. Although membrane lipid composition was not measured, we speculate that responses to different dietary lipids may have been mediated through changes in the production of lipid-derived, proinflammatory compounds.

Although differences in rectal temperature were observed during trial 4, body-surface temperature profiles were variable and not different among dietary treatments. During rising fever, peripheral vasoconstriction decreases heat loss by reducing dermal blood flow to accommodate a rise in core body temperature (Eiger and Kluger, 1983), thus allowing an upregulation of the infected host's immune response to fight off offending pathogens (Kluger, 1980). Blatteis et al. (1988) demonstrated that injection of LPS in sheep resulted in a rise of fever, whereas blood flow shifted away from heat-loss tissues (e.g., skin) to heat-production tissues. In the present research, it was expected that the injection of LPS would result in a rise in rectal temperature, and a concomitant decrease in body surface temperature, because of peripheral vasoconstriction and the shift in blood flow away from the skin.

Supplementation of diets with n-3 PUFA has been shown to alter the acute-phase protein response. Eicosapentaenoic acid was shown to downregulate the acute-phase protein C-reactive protein in human patients with pancreatic cancer cachexia (Wigmore et al., 1997). Barber et al. (1999) showed an increase in transferrin in pancreatic cancer patients with n-3 PUFA supplementation, and concluded that these fatty acids may help to stabilize the acute-phase protein response in such patients. Limaos et al. (1985) demonstrated that neither prostaglandins nor leukotrienes were involved in the production of haptoglobin and fibrinogen; rather, their biosynthesis was corticosteroid-dependent. This may suggest that FIB and HAP synthesis is independent of eicosanoid production and that differences in dietary PUFA intake are unlikely to influence their production. The lack of differences in FIB and HAP

stimulated by source of dietary lipid in the present studies may support this conclusion.

Several studies have determined the effects of feeding mice lipids with different fatty acid compositions upon the ability of stimulated immune cells to produce PGE_2 (Lokesh et al., 1988; Yaqoob and Calder 1995; Peterson et al., 1998). Overall, an inverse relationship between n-3 PUFA and PGE_2 production has been observed. Stated differently, as the ratio of n-3 to n-6 PUFA increases, a decrease in PGE production occurred. In the present studies, however, plasma levels of PGE were measured, not immune cell production of PGE. Our observation was that plasma concentrations of PGE (Tables 5, 7, and 9) were highly variable and not different among cattle fed the 3 dietary treatments.

Given the lack of dietary treatment effect on rectal temperatures in trial 5, we speculate that previous dietary treatment (trial 4) potentially resulted in a residual effect on the response to LPS. Endres et al. (1989) observed that cytokine production by human mononuclear cells was suppressed as late as 10 wk after n-3 supplementation was discontinued, indicating a long persistence of biochemical changes associated with n-3 supplementation. Therefore, the 60-d interval between trials 4 and 5 may not have been long enough to remove residual effects of PUFA supplementation. In addition, length of acclimation to the dietary lipid may have not been long enough to fully alter lipid membrane composition and, therefore, change inflammatory response. Pomposelli et al. (1989) also noted the length of acclimation to dietary fat as affecting fever response. In agreement with trial 4, results of trial 5 showed that changes in, and severity of, rectal temperature responses were more pronounced after the d 14 LPS injection, which is likely attributable to the animal's development of tolerance to the LPS challenge. Furthermore, body surface temperatures were highly variable and not different among dietary treatments.

Our data suggest that feeding full-fat soybeans to receiving cattle can depress feed intake and growth performance. This is in contrast to observations of Felton and Kerley (2000), who showed no detrimental effects on growth performance of finishing feedlot steers with the inclusion of up to 24% raw soybeans in the diet. Although the nature of this difference is not certain, we speculate that dietary changes in lipid source may interact with immune response in immunocompromised animals.

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